

| Ref # | Hits | Search Query  | DBs                           | Default Operator | Plurals | Time Stamp       |
|-------|------|---|-------------------------------|------------------|---------|------------------|
| L1    | 1    | ("6734023").PN.   | USPAT; EPO                    | OR               | OFF     | 2005/12/16 09:23 |
| L2    | 1    | ("6770486").PN.   | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:17 |
| L3    | 19   | stability same denaturant same (receptor or ligand or antibody) | US-PGPUB; USPAT; EPO; DERWENT | OR               | OFF     | 2005/12/16 10:19 |
| L4    | 5    | I3 and (MS or (mass adj1 spectrum\$))                           | US-PGPUB; USPAT; EPO; DERWENT | OR               | OFF     | 2005/12/16 10:19 |
| L5    | 75   | stability same urea same (receptor or ligand or antibody)       | US-PGPUB; USPAT; EPO; DERWENT | OR               | OFF     | 2005/12/16 10:19 |
| L6    | 18   | I5 and (MS or (mass adj1 spectrum\$))                           | US-PGPUB; USPAT; EPO; DERWENT | OR               | OFF     | 2005/12/16 10:21 |
| L7    | 5    | I4 and (MS or (mass adj1 spectrum\$))                           | US-PGPUB; USPAT; EPO; DERWENT | OR               | OFF     | 2005/12/16 10:23 |
| L8    | 7529 | (436/173,86,89,501,518,811). CCLS.                              | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:24 |
| L9    | 3    | I8 and I3   | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:25 |
| L10   | 1    | I8 and I4   | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:25 |
| L11   | 4    | I8 and I5   | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:25 |
| L12   | 9    | (fold\$ same denaturant) same ligand                            | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:25 |
| L13   | 2    | I12 and MS  | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:26 |
| L14   | 2    | I12 and (MS or (mass adj1 spectrum))                            | USPAT; EPO                    | OR               | OFF     | 2005/12/16 10:26 |

=> hydrogen exhange and interaction and mass

L1 0 FILE AGRICOLA  
L2 0 FILE BIOTECHNO  
L3 0 FILE CONFSCI  
L4 0 FILE HEALSAFE  
L5 0 FILE IMSDRUGCONF  
L6 0 FILE LIFESCI  
L7 0 FILE PASCAL

TOTAL FOR ALL FILES

L8 0 HYDROGEN EXHANGE AND INTERACTION AND MASS

=> deuterium and urea and ligand

L9 0 FILE AGRICOLA  
L10 1 FILE BIOTECHNO  
L11 0 FILE CONFSCI  
L12 0 FILE HEALSAFE  
L13 0 FILE IMSDRUGCONF  
L14 0 FILE LIFESCI  
L15 1 FILE PASCAL

TOTAL FOR ALL FILES

L16 2 DEUTERIUM AND UREA AND LIGAND

=> dup rem

ENTER L# LIST OR (END):l16

DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF'.

ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE

PROCESSING COMPLETED FOR L16

L17 2 DUP REM L16 (0 DUPLICATES REMOVED)

=> d l17 ibib abs total

L17 ANSWER 1 OF 2 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on

STN

ACCESSION NUMBER: 2000-0507759 PASCAL

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TITLE (IN ENGLISH): Origin of soft spin structure in Mn formate di-  
urea

AUTHOR: KUBO Hidenori; ZENMYO Kazuko; TOKITA Masahiko;  
MATSUMURA Manabu; TAKEDA Kazuyoshi; OOHASHI Tooru;  
YAMAGATA Kazuo

CORPORATE SOURCE: Fukuoka Institute of Technology, Wajiro, Fukuoka  
811-0295 Fukuoka 813-8581, Japan; Department of

Applied Physics, Faculty of Engineering, Kyushu  
University, Hakozaki, Fukuoka 813-8581, Japan; College  
of Science and Engineering, Iwaki Meisei University,  
Iwaki, Fukushima 970-8044, Japan

SOURCE: Journal of the Physical Society of Japan, (2000),  
69(8), 2669-2674, 8 refs.

ISSN: 0031-9015 CODEN: JUPSAU

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: Japan

LANGUAGE: English

AVAILABILITY: INIST-149, 354000092639230470

AN 2000-0507759 PASCAL

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AB Proton NMR measurements at low temperature show that Mn moment direction in Mn formate di-urea easily changes by temperature as well as the replacement of hydrogen with deuterium atom in urea

. The change in the moment direction is due to the competition of the magnetic anisotropy caused by the dipole interaction with the anisotropy caused by the surrounding ligand ions. The observed spin structure of four-fold screw rotations of antiferromagnetic layers is well understood by very small interlayer exchange interactions and magnetic anisotropies. The possibility is pointed out that the dipole interaction between the first nearest neighboring layers is the important origin of the transition to the three-dimensional long range order of this compound.

L17 ANSWER 2 OF 2 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on  
STN

ACCESSION NUMBER: 2000:30060770 BIOTECHNO

TITLE: Thermodynamic modulation of light chain amyloid fibril formation

AUTHOR: Kim Y.-S.; Wall J.S.; Meyer J.; Murphy C.; Randolph T.W.; Manning M.C.; Solomon A.; Carpenter J.F.

CORPORATE SOURCE: J.F. Carpenter, School of Pharmacy, Box C238, Univ. of Colorado Hlth. Sci. Center, 4200 E. Ninth Ave., Denver, CO 80262, United States.  
E-mail: john.carpenter@uchsc.edu

SOURCE: Journal of Biological Chemistry, (21 JAN 2000), 275/3  
(1570-1574), 39 reference(s)  
CODEN: JBCHA3 ISSN: 0021-9258

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2000:30060770 BIOTECHNO

AB To obtain further insight into the pathogenesis of amyloidosis and develop therapeutic strategies to inhibit fibril formation we investigated: 1) the relationship between intrinsic physical properties (thermodynamic stability and hydrogen-deuterium (H-D) exchange rates) and the propensity of human immunoglobulin light chains to form amyloid fibrils in vitro; and 2) the effects of extrinsically modulating these properties on fibril formation. An amyloid-associated protein readily formed amyloid fibrils in vitro and had a lower free energy of unfolding than a homologous nonpathological protein, which did not form fibrils in vitro. H-D exchange was much faster for the pathological protein, suggesting it had a greater fraction of partially folded molecules. The thermodynamic stabilizer sucrose completely inhibited fibril formation by the pathological protein and shifted the values for its physical parameters to those measured for the nonpathological protein in buffer alone. Conversely, urea sufficiently destabilized the nonpathological protein such that its measured physical properties were equivalent to those of the pathological protein in buffer, and it formed fibrils. Thus, fibril formation by light chains is predominantly controlled by thermodynamic stability; and a rational strategy to inhibit amyloidosis is to design high affinity ligands that specifically increase the stability of the native protein.

=> stability and denaturant

L18 33 FILE AGRICOLA  
L19 319 FILE BIOTECHNO  
L20 3 FILE CONFSCI  
L21 0 FILE HEALSAFE  
L22 0 FILE IMSDRUGCONF  
L23 209 FILE LIFESCI  
L24 107 FILE PASCAL

TOTAL FOR ALL FILES

L25 671 STABILITY AND DENATURANT

=> l25 and mass

L26 3 FILE AGRICOLA  
L27 17 FILE BIOTECHNO  
L28 0 FILE CONFSCI  
L29 0 FILE HEALSAFE  
L30 0 FILE IMSDRUGCONF  
L31 11 FILE LIFESCI  
L32 8 FILE PASCAL

TOTAL FOR ALL FILES

L33 39 L25 AND MASS

=> l33 and ligand

L34 0 FILE AGRICOLA  
L35 0 FILE BIOTECHNO  
L36 0 FILE CONFSCI  
L37 0 FILE HEALSAFE  
L38 0 FILE IMSDRUGCONF  
L39 0 FILE LIFESCI  
L40 0 FILE PASCAL

TOTAL FOR ALL FILES

L41 0 L33 AND LIGAND

=> l33 and interaction

L42 2 FILE AGRICOLA  
L43 5 FILE BIOTECHNO  
L44 0 FILE CONFSCI  
L45 0 FILE HEALSAFE  
L46 0 FILE IMSDRUGCONF  
L47 4 FILE LIFESCI  
L48 2 FILE PASCAL

TOTAL FOR ALL FILES

L49 13 L33 AND INTERACTION

=> dup rem

ENTER L# LIST OR (END):l49

DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF'.

ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE  
PROCESSING COMPLETED FOR L49

L50 8 DUP REM L49 (5 DUPLICATES REMOVED)

=> l50 and urea

L51 2 S L50  
L52 1 FILE AGRICOLA  
L53 5 S L50  
L54 1 FILE BIOTECHNO  
L55 0 S L50  
L56 0 FILE CONFSCI  
L57 0 S L50  
L58 0 FILE HEALSAFE  
L59 0 S L50  
L60 0 FILE IMSDRUGCONF  
L61 0 S L50  
L62 0 FILE LIFESCI  
L63 1 S L50

L64 0 FILE PASCAL

TOTAL FOR ALL FILES

L65 2 L50 AND UREA

=> d l65 ibib abs total

L65 ANSWER 1 OF 2 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.

(2005) on STN

ACCESSION NUMBER: 2003:46219 AGRICOLA

DOCUMENT NUMBER: IND23332972

TITLE: Evidence that thermodynamic stability of papaya glutamine cyclase is only marginal.

AUTHOR(S): Azarkan, M.; Amrani, A.; Zerhouni, S.; Oberg, K.A.; Ruysschaert, J.M.; Wintjens, R.; Looze, Y.

AVAILABILITY: DNAL (381 B524)

SOURCE: Biopolymers, Dec 5, 2002. Vol. 65, No. 5. p. 325-335

Publisher: New York, N.Y. : John Wiley & Sons, Inc.

CODEN: BIPMAA; ISSN: 0006-3525

NOTE: Includes references

PUB. COUNTRY: New York (State); United States

DOCUMENT TYPE: Article

FILE SEGMENT: U.S. Imprints not USDA, Experiment or Extension

LANGUAGE: English

AB Papaya glutamine cyclase (PQC), a glycoprotein with a molecular mass of 32,980 Da, is a minor constituent of the papaya latex protein fraction. In neutral aqueous solutions, PQC adopts an all-beta conformation and exhibits high resistance to both proteolysis and denaturation. Complete unfolding of PQC requires a combination of an acidic medium and chemical denaturant such as urea or guanidine hydrochloride. The unfolding process takes place through formation of an intermediate A state that accumulates in the absence of chemical denaturants and displays all the features of a molten globule state. The different conformational states--N (native), A (acid-inactivated), and U (unfolded)--have been characterized by means of circular dichroism measurements, fluorescence spectroscopies, Stokes radii determinations, and 8-anilino-1-naphthalenesulfonic acid (ANS) binding characteristics. The unfolding pathways of the enzyme was further studied to estimate thermodynamic parameters characterizing both transitions N reversible reaction A and A reversible reaction U. In its A state, PQC is catalytically inefficient and highly susceptible to proteolysis. Also, its thermodynamic stability is decreased by some 3-5 kcal/mol.

Conversion of the native to the A state involves digging up of five amino functions together with protonation of four to five acidic groups with

pK<sub>a</sub>s, in the native state, around 2.7. It proceeds both cooperatively and reversibly although, *in vitro*, the refolding process is slow. Unfolding of the A state, on the other hand, occurs with a low degree of cooperativity. The intermediate A state thus seems to be only marginally more stable than the unfolded state. The role of suspected internal ion pairs in the stabilization of the native state of this enzyme is discussed.

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ACCESSION NUMBER: 1993:23225653 BIOTECHNO

TITLE: Effect of pH and denaturants on the folding and stability of murine interleukin-6

AUTHOR: Ward L.D.; Jian Guo Zhang; Checkley G.; Preston B.; Simpson R.J.

CORPORATE SOURCE: Ludwig Institute for Cancer Research, P.O. Royal Melbourne Hospital, Parkville, Vic. 3050, Australia.

SOURCE: Protein Science, (1993), 2/8 (1291-1300)

CODEN: PRCIEI ISSN: 0961-8368

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 1993:23225653 BIOTECHNO

AB The conformation and stability of a recombinant mouse interleukin-6 (mIL-6) has been investigated by analytical ultracentrifugation, fluorescence spectroscopy, urea-gradient gel electrophoresis, and near- and far- ultraviolet circular dichroism. On decreasing the pH from 8.0 to 4.0, the tryptophan fluorescence of mIL-6 was quenched 40%, the midpoint of the transition occurring at pH 6.9. The change in fluorescence quantum yield was not due to unfolding of the molecule because the conformation of mIL-6, as judged by both urea-gradient gel electrophoresis and CD spectroscopy, was stable over the pH range 2.0-10.0. Sedimentation equilibrium experiments indicated that mIL-6 was monomeric, with a molecular mass of 22,500 Da over the pH range used in these physicochemical studies. Quenching of tryptophan fluorescence (20%) also occurred in the presence of 6 M guanidine hydrochloride upon going from pH 7.4 to 4.0 suggesting that an amino acid residue vicinal in the primary structure to one or both of the two tryptophan residues, Trp-36 and Trp-160, may be partially involved in the quenching of endogenous fluorescence. In this regard, similar results were obtained for a 17-residue synthetic peptide, peptide H1, which corresponds to an N-terminal region of mIL-6 (residues Val-27-Lys-43). The pH-dependent acid quenching of endogenous tryptophan fluorescence of peptide H1 was 30% in the random coil conformation and 60% in the presence of  $\alpha$ -helix-promoting solvents. Replacement of His-33 with Ala-33 in peptide H1 alleviated a significant portion of the

pH-dependent quenching of fluorescence suggesting that the interaction of the imidazole ring of His-33 with the indole ring of Trp-36 is a major determinant responsible for the quenching of the endogenous protein fluorescence of mIL-6.